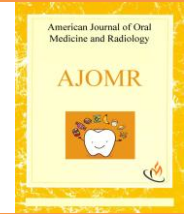




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TRIGEMINAL NEURALGIA- A REVIEW ARTICLE

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ABSTRACT

Neuralgia may be defined as proximal, intense, intermittent pain that is usually confined to specific nerve branches of head and neck (without any sensory or motor loss). Trigeminal neuralgia is also called TIC Dolo reux, trifacial neuralgia, fother gills disease and is the most common of cranial neuralgias and involves 5th nerve i.e trigeminal nerve.

INTRODUCTION

Trigeminal neuralgia has long been recognized in the medical literature; in fact, it was described as early as the first century AD in the writings of Aretaeus. It was later discussed by Johannes Baush in 1672. Nicolas Andre in 1756 used the term tic douloureux (painful spasm) to describe the disorder. Fothergill provided a vivid description of trigeminal neuralgia in 1773. Early treatments included bloodletting and application of bandages containing arsenic, mercury, hemlock, cobra and bee venom, and other poisons [1,2].

Functional Considerations of Trigeminal Nerve: The trigeminal nerve, the fifth cranial nerve, is largest cranial nerve, which contains both sensory and motor fibers. General somatic afferent fibers convey both exteroceptive and proprioceptive impulses.

Exteroceptive impulses of touch, pain and thermal senses are transmitted from the skin of the face and forehead; mucous membrane of nasal cavities, oral cavity, nasal sinuses, floor of the mouth, the teeth, the anterior 2/3rd of the tongue, the extensive portions of the cranial dura.

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Proprioceptive impulses [deep pressure and kinesthesia] are conveyed from the teeth periodontium, hard palate and temporomandibular joint receptors. The nerve is also involved in conveyed afferent fibers from stretch receptors in the muscles of mastication, the tensor tympani and the tensor veli palatini muscles, muscles of eye and the facial muscles. The trigeminal nerve is attached to the lateral part of the pons by its two roots, motor and sensory the two roots enter the middle cranial fossa.

Epidemiology

Trigeminal neuralgia is rare and thus statistical data regarding it scant. Early literature suggested a strong preponderance in women; however, current data indicate that only approximately 60% of the patients with trigeminal neuralgia are female. The annual incidence for women is approximately 5.9 cases per 100,000 women; for men it is approximately 3.4 cases per 100,000 men. Incidence increases with age. Although peak onset occurs between age 50 and 70 years, the disorder can also occur in children .the youngest child reported to have trigeminal neuralgia was approximately age 12 months; other children between age 3 and 11 years have also experienced the condition. No known racial or ethnic risk factors exist. Patient with multiple sclerosis may develop trigeminal neuralgia as a secondary symptom. However



this occurrence is relatively rare, involving only approximately 1% of patients with multiple sclerosis [3].

Clinical Features

With respect to classic trigeminal neuralgia, the pain occurs in short spasmodic episodes, often described as being similar to electric shocks. A typical attack lasts only a few seconds. However, subsequent attacks can follow within minutes. At its worst, the pain is completely paralyzing. It usually seems much localized within the area of the trigeminal nerve and does not radiate into other areas. The pain almost always affects only one side of the face. The area of the face affected reflects the branch of the trigeminal nerve that is involved. Trigger points, or areas of the face that with light pressure will trigger a pain attack, are a characteristic features of trigeminal neuralgia. Such points may be located on the lips, on the side of the jaw, underneath the eye, in the eyelid, or anywhere the trigeminal nerve reaches. There are several activities that can trigger an attack. Eating can become almost impossible, and loss of weight is common among those with the disorder. Shaving, applying makeup, and even talking can become difficult. In some cases, even a gust of wind can be enough to start an attack. An attack can also start without provocation [4].

Even without treatment, there can be periods of remission when pain is completely absent. This period-which can last days, week, months or even years-are unpredictable? However, without medical treatment, the pain usually returns.

Many patients with trigeminal neuralgia have symptoms that do not conform to those of classic trigeminal neuralgia. In addition to stabbing, shock-like pain, many patients experience pain that they describe as throbbing, burning, crushing or pulsating. For some, there is no remission from the pain. These atypical forms of trigeminal neuralgia are often very difficult to treat.

TYPES: There are 3 types of trigeminal pain: typical, atypical, and transitional.

Typical: The pain is sharp, like an ice pick or a shock. The pain comes and goes for short or long periods of time. The pain disappears for short or long periods of time. The pain has triggers that you can identify.

Atypical: The pain can be burning, aching, or throbbing, as well as sharp or stabbing. The pain may or may not disappear for periods of time. The pain has no known triggers.

Transitional: The pain is both typical and atypical. Trigeminal neuralgia can be classified as classic trigeminal neuralgia when it is not associated with an underlying neurologic disease or symptomatic trigeminal neuralgia when no neurologic disorder can be detected.

Trigeminal Neuralgia: Clinical Diagnostic Criteria:

1. **Character:** Discharge, electric shock, excruciating, superficial

2. **Intensity:** Moderate to very severe

3. **Duration:** Each pain episode does not last more than 2 minutes, several episodes during the day

4. **Periodicity:** Periods of weeks, months without pain, but also painless periods between attacks

5. **Site:** Trigeminal nerve distribution area, in general unilateral

6. **Irradiation:** Within trigeminal nerve area or beyond

7. **Triggering factors:** Innocuous stimulations such as eating, talking, bathing

8. **Relief factors:** Frequently sleep, anticonvulsant drugs

9. **Associated factors:** Triggering zones, weight loss, low quality of life, depression.

IHS classification suggests that at least four of these should be present for the diagnosis to be made [5].

Diagnosis

No medical test exists that can be used to clearly diagnose all cases of trigeminal neuralgia. However, establishing the diagnosis is usually not difficult, especially in cases of classic trigeminal neuralgia in which the symptoms are clear and distinct. A thorough medical examination should be performed, and a history of symptoms should be obtained. A recent study demonstrated that an examination alone was often insufficient to distinguish symptomatic from classic TN and that electrophysiologic testing of trigeminal reflexes was much more accurate. Local anesthetic blocks, which temporarily eliminate the trigger zone, may also be helpful in diagnosis. Since 10% of TN cases are caused by detectable underlying pathology, Medical test should be performed to rule out any serious medical problems; these tests can include computed tomography and magnetic resonance Imaging. Conventional MRI scan used to rule out the presence of a brain tumor or multiple sclerosis as a cause of a patient's facial pain are not, however adequate to observe the trigeminal nerve or an associated blood vessel. Fortunately, an improved form of magnetic resonance neuro imaging now makes it possible to observe both the nerve and associated blood vessels. The technique, called 3-D volume acquisition, is performed with contrast injection and uses thin cuts without gaps [6]. This technique is similar to MRI angiography and venography. The trigeminal nerve is easily observed in the axial plane when the MRI series is centered at the midpoint of the fourth ventricle. To ensure adequate evaluation, the nerve should be observed on 3 adjacent cuts. Early studies indicate that when an offending vessel is present, it will be detected 80% of the time.

Treatment

Initially, administration of anticonvulsant drugs was the treatment of choice for trigeminal neuralgia. There are now a variety of other effective treatments, both pharmacologic and surgical [7,8]. However, none of them is a cure.



Pharmacological Treatments

Carbamazepine: traditionally, carbamazepine, an anticonvulsant medication, has been used a first line drug for the treatment of trigeminal neuralgia. In fact, some clinicians believe that if orofacial pain does not respond to carbamazepine, then it is not trigeminal neuralgia pain.

Delivery Systems for the Orofacial Region

The purpose of a local delivery system is to apply a medication for a therapeutic action in a site specific manner. The drug's molecular structure and its pharmacological behavior dictate the delivery site and system. Use of topical medications in the orofacial region is accompanied by some inconveniences. For example, when applied intra orally (such as in a dissolving lozenge), these agents will dissolve in saliva and consequently spread throughout the mouth and down the throat. If the topical agent does have some muco-adhesive properties—in other words, if it is a gel or cream—it can be painted on the appropriate site, but, again, it will wash away from the area quickly. Several forms have been developed to counter this problem: toothpaste; chewing gum; candy; adhesive patches and powders; dissolving tablets, lozenges and lollipops; tissue-covering stents; dissolving polymeric devices; mouthwashes; and medicated lipsticks.

Muco-adhesive bases: Several muco-adhesive bases and oral pastes allow medications to be delivered to the intraoral and extra oral tissues with great specificity. One common muco-adhesive base that is very useful for oral neuropathic pain is available over the counter already compounded with a local anesthetic agent (for example, in Orabase- B, which contains 20% benzocaine). This muco-adhesive base remains in place for several hours after application. It also is used frequently as a carrier vehicle for applying other medications (such as capsaicin) to the oral mucosa.

Transdermal creams: Recently, a new medication vehicle preparation has been developed that provides rapid dermal and mucosal penetration and lends itself to being compounded with other medications. The transdermal cream is a pluronic mixture of lecithin and organogel. The organogel provides a hydrophilic vehicle for binding and carrying the admixed medication, while the lecithin increases the vehicle's ability to penetrate the lipophilic epidermal barrier. Changing the ratio of lecithin to organ gel moderates the cream's solubility through the lipophilic membrane.

Toothpastes: Standard toothpaste also can be used as a vehicle for local delivery, and compounding pharmacies have medications—such as lidocaine—for neuropathic pain available in a dental paste form. As a delivery vehicle, paste has advantages in that it incorporates a medication protocol in the patient's daily routine, which may increase his or her compliance with therapy.

However, no studies have shown the efficacy of this vehicle.

Medicated chewing gum and candy: The use of other systems such as medicated candies and chewing gum can provide the possibility of implementing a slow-release therapy or a gradient in the concentration of different medications. Oral capsaicin has been used in a hard-candy vehicle, producing significant but temporary pain reduction in patients with oral mucositis resulting from cancer therapy. Local anesthetics also have been compounded as a lollipop candy, for a continuous delivery in the oral mucosa. The technique is not well-developed yet and needs to be studied. It is still unclear if the response to these delivery systems may be achieved through systemic delivery, rather than a local effect or combination, and additional research is needed in this arena.

Dissolvable tablets and lozenges: The use of dissolvable tablets or lozenges is another way to achieve a slow delivery of medications to the oral mucosa. In a study with dissolvable tablets, it was shown that a drug released in the upper buccal sulcus would migrate in an anterior direction. A tablet placed in the lower buccal sulcus showed a larger local concentration of the drug around the delivery system, and the widest distribution of medication was achieved with the tablet placed under the tongue.

Mouthwashes: A mouthwash can be held in the oral cavity for up to several minutes without significant discomfort. It then may be swallowed or ejected (the latter if one wishes to reduce the potential of systemic drug delivery). The main use for mouthwashes is oral hygiene, reduction of plaque formation and control of stomatitis. The potential use of this system for neuropathic intraoral conditions is linked with its ability to reach a wide area of tissue with each application.

Medicated lipstick: When local delivery to the lips is indicated, a lipstick vehicle can be a suitable alternative. This system will allow easy application of the medication, and patients accept it readily. If an ectopic generator or trigger zone of a neuropathy is located in the lips, this might be an adequate therapeutic option.

Options of surgery: Surgery can be divided into two types, viz. restorative and destructive. The best surgical treatment is to remove the cause of pain. If it is due to vessel loop or tumour compression, surgical decompression should be the procedure of choice. When no cause is identified, or the cause is difficult to deal with, one can interrupt the nerve so that it cannot conduct the pain signal to the brain.

CONCLUSION: The presence of trigeminal sensory deficits, bilateral involvement, or abnormal trigeminal



reflexes is useful indicators of symptomatic TN, whereas younger age of onset, involvement of the first division, unresponsiveness to treatment, and abnormal trigeminal evoked potentials are not. Use of carbamazepine or oxcarbazepine is recommended as first choice pharmacological treatment in classical TN, and baclofen or lamotrigine as second choice. Although all the surgical procedures are inherently supported by low-level evidence, the results in thousands of patients indicate that the surgical treatments for trigeminal neuralgia are

efficacious and acceptably safe. An evidence-based direct comparison between the different surgical procedures is so far impossible. To briefly differentiate them, however, we may summarize that the percutaneous Gasserian lesions can be safely performed in the elderly but often engender facial numbness, microvascular decompression provides the longest-lasting pain relief but involves some risk of major neurological complications, gamma-knife is the least invasive and safest procedure but pain relief may take one month to develop [9].

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